LETTER TO EDITOR

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Nephrotic syndrome in plasma cell leukaemia - A rare presentation

M Diogo, C Faustino, T Pimentel, J Amorim, i Tavares
Departamento Medicina Interna Hospital São Marcos, 4700 Braga, Portugal

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Correspondence Address:
M Diogo
Departamento Medicina Interna Hospital São Marcos, 4700 Braga
Portugal
Login to access the email ID

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Sir,

Being less than 5% of all plasma cell disorders, most publications on plasma cell leukaemia (PCL) are based on case reports. In 2005 a 57- year- old man was admitted to our institution because of edema and dyspnea. He had a 1- month history of fatigue, anorexia, weight loss of 8 kg and a sharply delimited dorsal pain. Physical examination revealed pallor, hepatomegaly and edema. Laboratory data were as follows: hemoglobin 9.9 g/dl, white blood cell count 10.7×10^{9} /l with 65% plasmacytoid lymphocytes, urea

of 580 mg/l and creatinine of 34 mg/l. Calcium was elevated as well as β_2 - microglobulin (26144 ng/ml). There was no M- component. A skeletal bone survey showed an osteolytic lesion at the dorsal spine with fracture and small lesions at the lumbar spine. Ultrasonography revealed no renal alterations. Urine protein electrophoresis showed κ light chains 0.0068 g/l and λ light chains 3.91 g/l; 24- h urine collection revealed 12 gr of proteins. A bone marrow examination revealed hypercellularity with a 70% infiltration of plasma cells and a blood smear with a 24% infiltration, which were CD38- positive, CD 138- positive and CD20- negative. The DNA content study showed hyperploidy. The renal biopsy revealed a massive infiltration of the tubulointerstitial compartment by plasma cells and heavy deposition of l- light chains [Figure 1] with no amyloid substance. We administered a VAD regimen on days 1- 4, 9- 12 and 17- 20 at 4- week intervals.

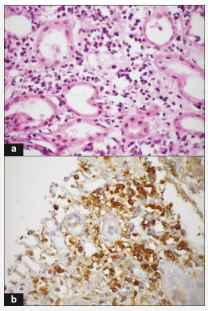

Figure 1: Kidney biopsy showing a diffuse infiltrate of atypical plasma cells mainly at the tubulointerstitial compartment (hematoxylin- eosin stain, original magnification × 400 , a) and λ light chains (b)

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Despite aggressive chemotherapy regimen, response to treatment is still poor and successful therapeutic regimen has not been yet established. [1],[2] Our patient was treated following the VAD regimen having received five courses at 4- week intervals without hematological remission. The patient died from tumor progression 4 months post -diagnosis.

Up till now, immunophenotypic studies have shown that myeloma cells from PCL characteristically do not express CD56. [3] These data lead some authors to consider the absence of CD56 as a hallmark of PCL and that could discriminates it from MM. Here relies a striking difference of our report were the CD56- positivity is not in agreement with the vast majority of the medical literature (most works report CD56 - negativity). Very few cases have studied DNA cell content in their reports; the vast majority of cases have a DNA index of 1 or less. [4] Here is another difference since our patient displayed hyperdiploidy.

The hematological malignancies associated with nephrotic syndrome are mainly due to Hodgkin and non- Hodgkin's lymphoma and chronic lymphocytic leukemia. Renal disease occurs in up to 50% of patients with MM and sometimes, more than one is seen. Being multifactorial, renal dysfunction attributable to leukemic infiltration is rare and mainly described for chronic lymphocytic leukaemia. [5] To the best of our knowledge no case of such massive kidney infiltration by plasma cells has previously been reported. Renal failure has the potential to substantially jeopardize the chances of cancer patients receiving optimal treatment. The strategy has to consider both plasma cell clone and renal manifestations being necessarily a combined approach, which was not possible in our patient. [6]

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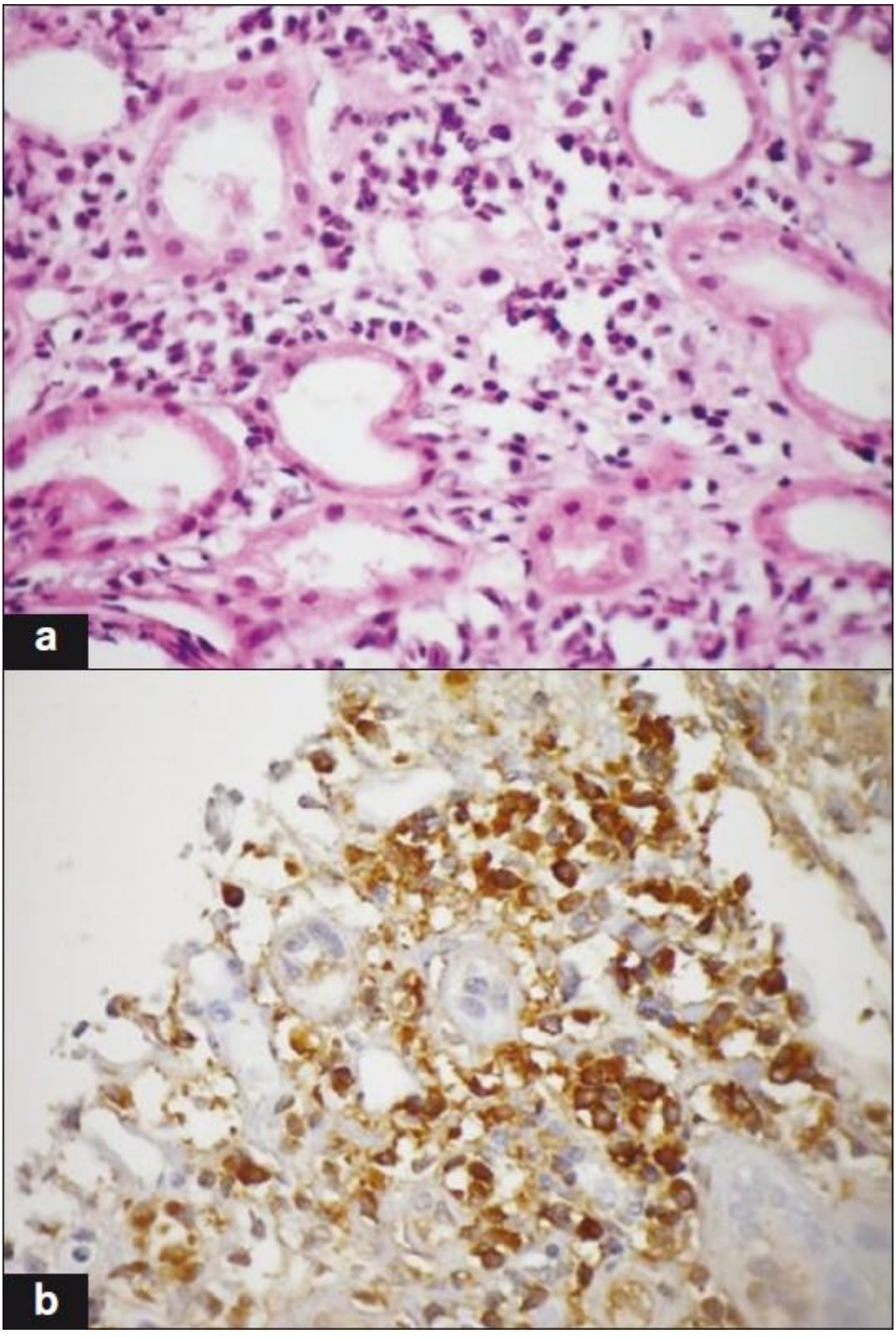
Figures

[Figure 1]

Figure 1: Kidney biopsy showing a diffuse infiltrate of atypical plasma cells mainly at the tubulointerstitial compartment (hematoxylin- eosin stain, original magnification × 400 , a) and λ light chains (b)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